

PALAEOPATHOLOGY OF HUMAN BONES FROM MURRAY RIVER REGION BETWEEN MILDURA AND RENMARK, AUSTRALIA

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The skeletal material has been meticulously described (Blackwood and Simpson, this *Memoir*) and little remains to be added. Unfortunately many of the skeletons are more or less fragmented and incomplete. This makes palaeopathological study more difficult. Nevertheless, some observations may be made comparing these Aboriginal bones with those held in the Murray Black Collection of the Anatomy Department, University of Melbourne (Professor L. J. Ray), and in the National Museum of Victoria (Mr John McNally).

As in the reference collections, none of the skulls in the present series shows evidence of cribra orbitalia, parietal hyperostosis, biparietal thinning or other congenital anomaly. The skull base of skeleton 59 is markedly asymmetric, but this is probably exaggerated by post-mortem distortion (Pl. 17, fig. 2). Similar asymmetry has been noted in the Murray Black Collection. Indeed, the only important congenital anomaly noted in the skeletons is fusion of thoracic vertebrae in skeletons 60 and 64 (Pl. 18, fig. 3-4). There is no evidence whatsoever that this fusion is of the acquired type due to degenerative spinal arthropathy.

There is no trace of mastoiditis, sinusitis, nor of any other recognizable inflammatory disease process. In particular, neither crania nor post-cranial bones show any changes attributable to treponemal disease (trepanarid) which is a conspicuous feature of Aboriginal bones studied by me in Melbourne, Canberra, Sydney and Adelaide, although not in Queensland Aboriginal skeletons studied in Brisbane. Further there is no evidence of degenerative joint disease of the osteo-arthritis type nor of pyogenic infection of bones or joints. The tibial periosteal reaction seen in skeleton 25 is almost certainly of traumatic origin (Pl. 18, fig. 1).

This is a not uncommon finding in skeletal collections from cultures of all degrees of antiquity. In contrast with the other reference collections in Melbourne there is little evidence in the present series of other trauma except for a probable healed fracture of the left clavicle of skeleton 60 sustained in early life (Pl. 18, fig. 2).

Skeleton 64 shows marked bowing of the radii (Pl. 18, figs. 5-6). This bowing of long bones has also been noted in the Murray Black Collection. The condition appears to be related to or analogous to 'boomerang tibia'. There is at present no evidence that the condition is related to trepanarid nor to malnutrition. It may well be of genetic origin.

As in the skulls held in the Murray Black Collection, and in the National Museum Collection, severe tooth wear is often evident. This is probably related to the nature of the diet, the admixture of sand or grit and possibly also to the oral manipulation of wooden artifacts. Mushrooming of the condyles of the mandibles, which is fairly frequent in the reference collections, has not been seen in the present series. Where wear has been severe it may be followed by root exposure, opening of the tooth pulps and apical infection. Some skulls show loss of teeth as in skeleton 37 where the mandible is largely edentulous (Pl. 17, fig. 3). Dental caries is *not* seen; it would appear to be very rare in undoubted pre-contact specimens of Aboriginal teeth.

Crowding of incisors as noted in the young adult skeleton 17 is not uncommon in Aboriginal skulls (Pl. 17, fig. 4). Occasionally, adventitious or impacted teeth are also noted as exemplified by that seen in the palate of skeleton 61 (Pl. 17, fig. 1). Similar observations have been made in the Murray Black

Collection and in the National Museum of Victoria.

It is probably unwise to generalize on these observations in view of the smallness of the sample and the damage and loss of bones in many of the skeletons. The dental appearances are similar to those seen in the Melbourne collections in skulls from around the Murray River with regard to degree of wear, absence of caries and minor anomalies. However, in contrast is the absence of evidence of major trauma, degenerative joint disease, pyogenic infection and trepanarid. Whether this is due to sampling or whether the group described here is in some way different will only be resolved by further skeletal collection from the area surveyed in this report.

It is intended to publish the study of palaeopathological changes in Aboriginal bones held in Melbourne and the other cities as a memoir.

References

- BROTHWELL, D. R. (ed.), 1963. *Dental Anthropology*. Pergamon Press, Oxford.
- and SANDISON, A. T. (editors), 1967. *Diseases in Antiquity*. Thomas, Springfield.
- CAMPBELL, T. D., 1925. *Dentition and Palate of the Australian Aboriginal*. Hassall Press, Adelaide.
- SANDISON, A. T. 1968. Pathological changes in the skeletons of earlier populations due to acquired disease and difficulties in their interpretation. In *The Skeletal Biology of Earlier Human Populations*, ed. D. R. Brothwell, Pergamon Press, Oxford.

Explanation of Plates

PLATE 17

- Fig. 1—Impacted tooth in palate. Skeleton 61.
 Fig. 2—Congenital asymmetry of skull base, probably exaggerated by post-mortem distortion. Skeleton 59.
 Fig. 3—Partially edentulous mandible, Skeleton 37
 Fig. 4—Crowding of incisor teeth. Skeleton 17.

PLATE 18

- Fig. 1—Tibial periosteal reaction due to trauma. Skeleton 25.
 Fig. 2—Healed fracture of clavicle sustained in early life. Skeleton 60.
 Figs. 3-4—Congenital fusion of two thoracic vertebrae. No evidence of acquired disease. Skeleton 64.
 Figs. 5-6—Bowling of radii, probably of genetic origin. Skeleton 64.

Photographs:

Plate 18, fig. 4. Sir Robert Blackwood
 Remainder, Edmund D. Gill





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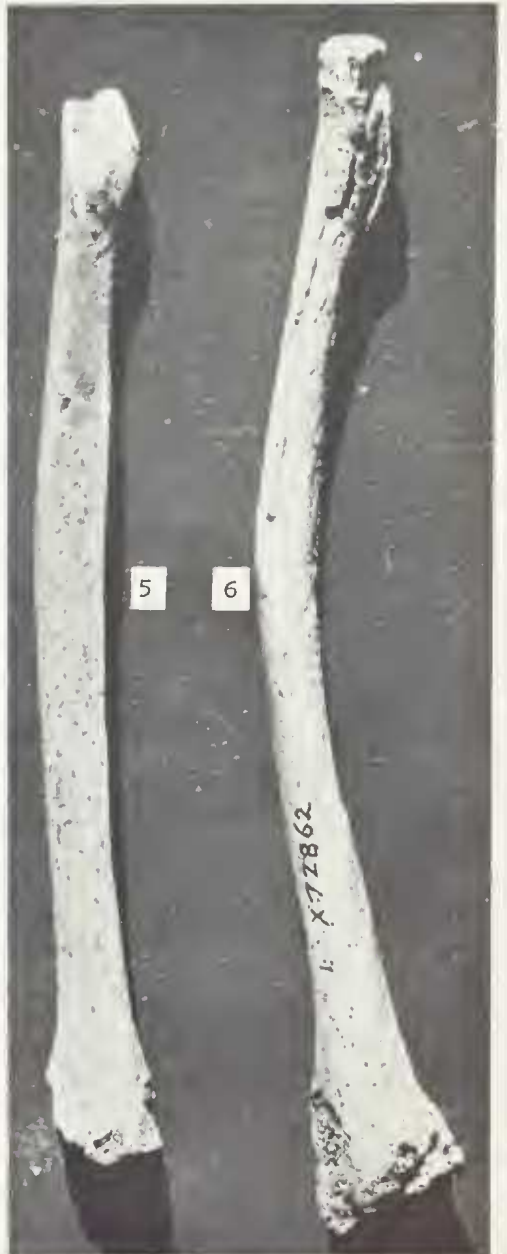
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